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In this issue of *Adipocyte*

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A Sensitive Issue pp 88–96

With the increasing prevalence of obesity in today's society, researchers have been looking into the role of white adipose tissue (WAT) and its role in insulin sensitivity and metabolism. A link between obesity and insulin sensitivity has been found, specifically regarding leptin, adiponectin, and adipolin. In this review by Knights et al., the roles of these adipokines are explored with the hopes of furthering our understanding of their involvement in insulin resistance and their potential ability to improve insulin sensitivity in the presence of obesity.

3T3-L1 Cells and the Chemokine Network pp 97–106

The low grade chronic inflammatory state that obesity presents is known to be one of the links between it and other diseases such as type 2 diabetes, heart disease, and cancer. It is also known that this kind of inflammation is associated with chemokine networks and the recruitment of immune cells. In this research paper by Kabir et al., the authors use 3T3-L1 cells in a differentiation model in order to observe the chemokine profiles during adipogenesis, as well as how it is changed by tumor necrosis factor-α (as a pro-inflammatory factor) and epidermal growth factor (as a growth factor) (**Fig. 1**). Their findings show an increase in CXCR2-mediated signaling in the model in the presence of growth factors like EGF when compared with proinflammatory factors like TNFα.

Adipose Tissue and Tumor Growth pp 107–14

Although the role of adipose tissue has previously only been thought of as inert energy storage, how it relates to the formation of breast tumors has yet to be fully explored. Sturtz et al. seek to shed some light on the role of adipose tissue in breast tumorigenesis. In this research paper, the authors compare gene expression data from adipose tissue both adjacent to and distant from invasive breast tumors and tissue adjacent to non-malignant tumors. The results showed increased expression of several anti-inflammatory genes in invasive tumor-adjacent adipose tissue, thus supporting the idea that increased immune tolerance in adipose tissue in invasive breast tumors may play a role in tumor growth.

Binge Eating and Lipolytic Function pp 115–20

In this brief report, authors Wiedemann et al. expand on their previous work and show how a short, four day high fat diet (HFD) can impact glucose tolerance and insulin sensitivity. By comparing chow-fed mice with those on a HFD, the authors determined that lipolysis remained unaltered in HFD-fed mice, and, these mice retained their insulin sensitivity. Unexpectedly however, free fatty acid concentrations were increased in HFD-fed mice leading the authors to conclude that short-term HFDs do not necessarily have an effect on the lipolytic function of adipocytes.

Short Chain Fatty Acids and Lipolysis pp 121–5

Although not quite understood, it has been reported that the process of lipolysis (hydrolysis of triacylglycerol into glycerol and non-esterified fatty acids [NEFA]) can be reduced by short chain fatty acids. Authors Aberdein et al. aimed to understand whether adipose tissue

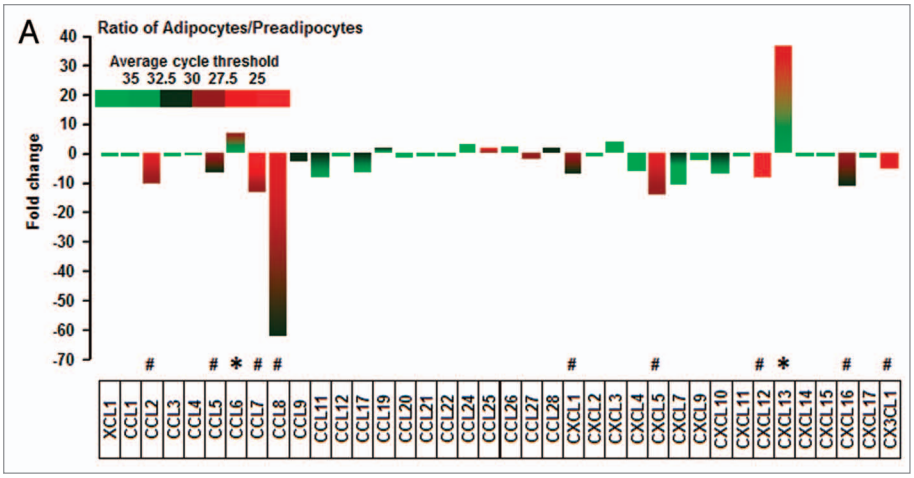

Figure 1. Figure detail from Kabir et al., p 98.

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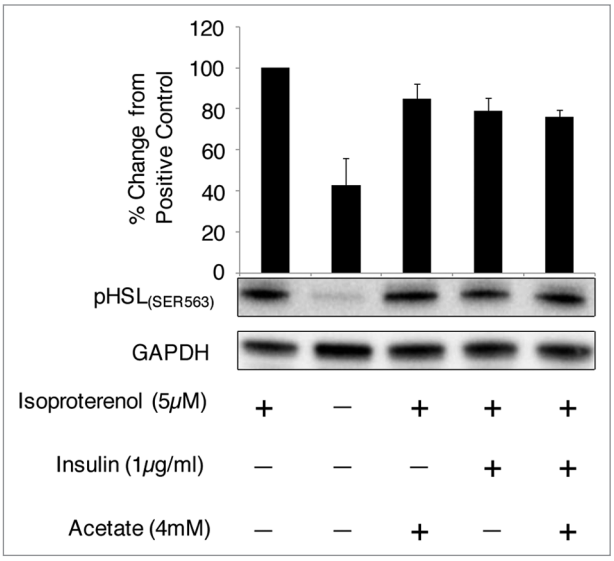

Figure 2. Figure from Aberdein et al., p 122.

lipolysis in vitro is affected by sodium acetate through the use of 3T3-L1 adipocytes. While levels of phosphorylation of HSL(SER563) in the adipocytes were reduced after being exposed to acetate, thus leading to a reduction in NEFA release, the acetate had no effect on glycerol release (**Fig. 2**).

Adipocytes and Endothelial Cells: Mediators of Adipose Inflammation pp 126–31

Adipose tissue is made up of more than just adipocytes and contains other cell types such as endothelial cells, and it is known that both adipocytes and endothelial cells produce a variety of inflammatory mediators. Authors Vielma et al. explore the ways in which immune cell production of cytokines are affected by mediators from adipocytes and endothelial cells in this brief report in order to observe the potential ability of these cells to serve as immune regulatory cells. Their study concluded that although when separate, adipocytes and endothelial cells serve to stimulate production of Th1, Th2, and most inflammatory cytokines and chemokines, when mixed they actually inhibited Th1 profile with sustained Th2-type inhibitory and inflammatory phenotype.

A Closer Look at DNA Methylation Profiles pp 132–40

Due to their roles as regulators of energy metabolism and insulin sensitivity, the adipokines leptin and adiponectin have appeared in a number of recent studies. Having recently reported a correlation between placental DNA methylation levels at *LEP* and *ADIPOQ* (encoding for leptin and adiponectin) gene loci and maternal glucose concentrations, the authors of this brief report compared DNA methylation in the same gene loci in blood and adipose tissues in obese class III subjects. What results is the first study to compare *LEP* and *ADIPOQ* DNA methylation levels in adipose tissues to those in blood to determine if DNA methylation of blood cells reflects that of adipose, and, if such studies can predict susceptibility to obesity and metabolic disease in later life.

Immunometabolism and Yo-Yo Dieting pp 141–5

Due to recent research, the field of immunometabolism has expanded greatly, to the point where adaptive immunity as well as innate immunity is looked at as being closely involved with obesity and associated metabolic dysregulation. This commentary by Anderson-Baucum et al. builds on previous research where the authors reported increased T-cell accumulation in adipose tissue as a result of weight cycling in mice. Here they expand on the possibility that this T-cell increase may actually represent a local secondary immune response to self-antigens exposed in adipose tissue during obesity (**Fig. 3**).

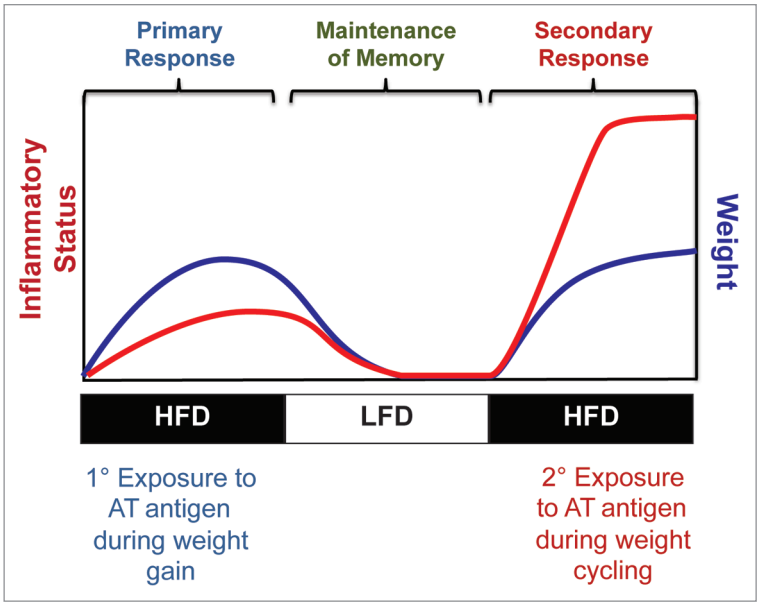

Figure 3. Figure from Anderson-Baucum et al., p 143.

The New AdipoChaser pp 146–50

Unsatisfied with current assessments of white adipose tissue life cycles and turnover rates due to technical limitations, authors Wang and Scherer present a new model dubbed the "AdipoChaser" mouse model. This commentary discusses the recent findings of this novel model in the tracking of adipogenesis in the presence of high fat dieting as well as cold exposure and β-3 agonist stimulation and offers a new understanding of adipose tissue development (**Fig. 4**).

Developmental Androgen Excess And The Metabolic Syndrome pp 151–4

This commentary by Mauvais-Jarvis explores the role of developmental androgen excess with regards to the programming of adult metabolic dysfunction. In a model in which male and female mice with neonatal exposure to testosterone were compared with control groups, it was shown that the exposure in females can lead to the development of features of metabolic syndrome similar to those observed in women with polycystic ovary syndrome. However, males with neonatal exposure exhibit the opposite features, with decreased lean mass and food intake.

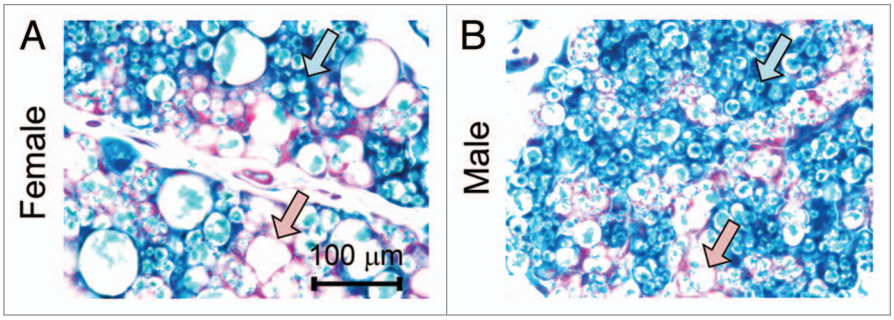

Figure 4. Figure from Wang and Scherer, p 149.

Seeing Brown pp 155–9

Although white adipose tissue is known as the driver of obesity and is most closely associated with metabolic disease, the importance of brown adipose tissue (BAT) is gaining recognition due to recent studies. Unfortunately, the identification and detection of BAT in adult humans has proved to be technically challenging. A new peptide probe that binds BAT vasculature that can be used to detect BAT in whole body imaging was recently reported by the author, and in this commentary, author Kolonin discusses several of the open questions needing to be addressed regarding this probe.

TIPping the Balance in Adipogenesis pp 160–5

The transcriptional cofactor and acetyltransferase Tip60 has been shown to be important in later stages of differentiation as well as some of the earlier steps in adipogenesis. With studies showing that protein levels of Tip60, but not its mRNA, are upregulated during adipogenesis as well as its degradation by the proteasome, authors Gao and Kalkhoven sought to discover whether Tip60 is stabilized through deubiquitination during adipogenesis. This commentary discusses their study, and looks at the role of Tip60 in cellular differentiation as well as the process of protein deubiquitination.